tunities and facilities for doing better work and to their communities a great reduction in health hazards.

Private hospitals of this class feel and accept their part of a community responsibility in the care of those unable to care for themselves, and are entitled to point with just pride to the fact that a goodly share of their earnings are paid in dividends of service to the less fortunate.

Unfortunately there are too many so-called private hospitals that have no such vision, and they may not be credited with the highest intentions or service. In some instances their privacy is used as a kind of smoke barrage to disguise incompetency or what not, which could not continue to propagate if hauled out into the open. Some of these so-called hospitals are dirty, badly designed firetraps without adequate equipment, personnel or other facilities for doing good work. Some of those better constructed and equipped make their work so coldly commercial that the patient seems to be an exotic in their wards.

When private profit-making hospitals are owned by physicians, there is danger of criticism that the thought of dividends may at times be influential in prolonging the stay of patients, unnecessary charges or the glossing over of poor service. There is no foundation for such criticisms in the majority of instances, any more than there is when a pharmacy is owned by a physician, but in both instances there exists the *opportunity* to criticize, and this is sufficient to insure a regrettable amount of it. Furthermore, there is just enough of truth in the situation in some hospitals to make it easy for all to be included by destructive critics.

On the other hand, there are undoubtedly certain advantages to physicians in being interested in their hospitals. The connection insures loyalty, co-operation and tangible assistance in the work of the hospital, not always easy to secure and hold when the only excuse for loyalty and co-operation consists in the opportunity to do a large amount of free work, often without adequate facilities and with no hope of recompense.

This phase of hospitals is discussed quite frankly because of frequent requests from physicians for an opinion upon the subject.

GOOD HOSPITAL ORGANIZATIONS.

Corporation management, with Boards of Directors representing stockholders, university, church, society or some other interest, constitutes the basic plan of the majority of good hospital organizations. This is the usual organization of business. Membership on the board of directors ought to be broad enough to satisfy hospital requirements as well as the various community interests, and the executive functions should be centralized as nearly as may be in the hands of one of its members, limiting the activities of the board as a whole to outlines of policy and questions of moment.

The principal difficulty with this form of organization is in non-attendance at meetings and lack of interested effort on the part of individual directors. It is an amazing fact, all too frequently seen, that members of hospital boards of directors give indifferent attention to their responsibilities, which may be many times greater economically and publicly than some small business of another type which will have their services, study and attention.

The advisability of physicians as members of hospital boards already is answered in the discussion of the private hospital organization. Public health officers and physicians not engaged in practice are especially valuable members of governing bodies and each community service hospital is benefited by numbering one or two upon its board of directors.

An exceedingly prevalent fault with hospital boards is, that they are too large. Seven members should be considered the maximum, unless the organization provides for an executive committee of three or five members with very extensive powers. Large boards with loose organization and numerous committees dealing with all sorts of hospital problems are no more suitable for a hospital than they would be for a bank or a department store. Such a body acting in an advisory capacity to a smaller administrative council has much to commend it in communities where the hospital function must be discharged to the satisfaction of a multiplicity of interests.

Original Articles

NEURO-OTOLOGY: ITS RELATION TO GENERAL MEDICINE.*

By FRED C. LEWITT, M. D., San Francisco.

Any investigation which adds to our knowledge of the function or anatomy of any part of the body is of value. When the knowledge thus obtained aids us clinically and can be applied with benefit to our patients, it becomes of the greatest importance, not to a few, in some special work, but to all. Of such value is the work that has been done in the investigation of the relation of the internal ear to the central nervous system. To this study has been given the name "Neuro-otology." This study has not only added to our knowledge of the anatomy and physiology of the internal ear in its relation to the central nervous system, but has also given us the means of obtaining data, valuable from a diagnostic point of view, in the examination of our patients.

While neuro-otology is primarily a study of the vestibular portion of the inner ear, and its pathways through the brain, its value, by the practical application of the data gained from the examinations, is being appreciated more and more both in medical and surgical diagnosis.

We know that the inner ear consists of two divisions which have separate functions—the cochlea which is concerned with hearing, and the static labyrinth which is concerned with equilibrium. The auditory function of the ear has been known since the beginning of medicine. It has only been within the last half of the last century that we have come to appreciate the role the internal ear plays as the chief organ of equilibrium.

It was through the work of such men as Purkinje, Flourens and Ewald that the relationship between the static labyrinth and equilibrium was first known. Later this work was further advanced by the work of Meniere. To Barany

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of Vienna however, goes the honor of placing the knowledge gained from this work in a position of practical and useful value to the otologist. But to Isaac H. Jones and those working with him in Philadelphia, goes the honor of working out the separate pathways of the canals through the brain stem, and so standardizing the methods of examination and so simplifying the interpretation of the results thus obtained that the work is no longer, to use a borrowed expression, "terra incognita."

We are all familiar with the two reactions resulting from the stimulation of the normal vestibular apparatus—nystagmus and vertigo. Past pointing is a secondary reaction dependent upon vertigo.

If each time the ear of a normal person is stimulated, these reactions appear in a definite manner depending upon the method of stimulation used, it must be because there are certain pathways from the semi-circular canals to the brain through which the stimulation must pass and the appearance of the reaction cannot be merely a coincidence. No more can we say it is a coincidence if after stimulation, one or both of these reactions fail to appear. The failure of any reaction to appear calls for an explanation by the examiner.

When we see a case with a normal nystagmus but no vertigo and past pointing, or a normal vertigo and past pointing but no nystagmus, we must conclude that there are *separate* pathways for these different reactions. Likewise when we obtain a reaction from the stimulation of the horizontal canal but none from the stimulation of the vertical canals or vice-versa, we must also conclude that there are *separate* pathways for the horizontal and vertical canals.

These separate pathways as worked out by Jones are as follows:

Fibers from the *horizontal* canal enter the 8th nerve as part of the vestibular portion of the nerve and continuing their course in the nerve enter the brain stem at the junction of the medulla and pons and pass to Deiter's nucleus. Here the fibers divide into a Y. One arm of the Y passes through the nucleus triangularis toward the median line to enter the posterior longitudinal bundle. This is the pathway for the nystagmus reaction from the horizontal canal. The other arm of the Y passes to the cerebellum by way of the *inferior peduncle* and terminates in the three cerebellar nuclei-nucleus glabosis, nucleus emboliformis and nucleus fastigii. A few fibers also pass to the dentate nucleus.

From the cerebellar nuclei the fibers reach the cerebrum by two pathways—the chief one being from the nuclei through the superior peduncle to the crura, through the decussation of the crura to the cerebral cortex of the opposite side terminating in the cortical center for vestibular impulses which is situated in the posterior portion of the first and second temporal convolutions. From here a few association fibers connect the center with other parts of the cerebrum particularly the frontal lobe. The second or minor pathway is from the superior peduncle to the crura to the temporal lobe of the same side. This, the vestibulo-cerebellar-cerebral tract, Jones calls the subjective circuit and it is the pathway for the vertigo and past pointing reactions.

(2) The fibers from the *vertical* canals entering the brain stem through the 8th nerve, pass mesad to the fibers of the horizontal canal, ascend through the medulla to the upper part of the pons where they also divide into a Y—one arm of the Y going directly to the posterior longitudinal bundle forming the vestibular-ocular tract for the vertical canals. The other arm of the Y passes through the *middle peduncle* to the cerebellar nuclei mentioned above. From here the fibers have the same pathway as those from the horizontal canal.

With this picture of the separate pathways clear in our mind, we can locate the site of the lesion if after stimulation any of the reactions which normally should be present fail to appear.

It is not within the scope of this paper to describe or explain these reactions. It is taken for granted that we are all familiar with them. However certain points may be mentioned briefly which will aid us in determining beforehand what the normal reaction should be.

In regard to nystagmus the slow or vestibular component of the nystagmus is always in the direction of the endo-lymph flow.

Vertigo—the second primary reaction due to stimulation of the normal labyrinth—is always in a direction opposite to the endo-lymph flow.

Past pointing is a secondary reaction due solely to vertigo, and is in a direction opposite to the vertigo. If there is no vertigo there will be no past pointing.

The most conspicuous symptom we have to deal with in this study of neuro-otology is *vertigo*. To the patient it is the *complaint* for which he seeks relief. Varying in intensity and duration, it at times becomes so distressing as to prevent the patient following out his normal pursuits and our treatment is valued only to the degree to which relief is afforded.

To the physician vertigo is a *symptom* which occurs very frequently and in numerous and various pathological conditions. Under such divers conditions does vertigo appear that little has been done in the way of investigation as to its exact cause and definite methods of diagnosis.

Vertigo is related to a disturbed equilibrium and in order to understand it and arrive at its cause we must first have a clear idea of the different factors which go to maintain equilibrium.

Equilibrium is maintained by a constant flow of impulses to the brain from the eyes, ears, and the muscles, tendons and joints, in other words from our deep muscle sense. Normally this flow of impulses is constant and so balanced or harmonized that we are not conscious of it. It is only when this balance is disturbed, resulting in a confusion and disturbance of equilibrium, that we experience vertigo. Of the sources of these afferent impulses maintaining equilibrium those from the vestibular portion of the inner ear are the most important. A lesion involving the vestibular apparatus anywhere in its course from

the end-organ through its pathways in the brain results in vertigo. As the maintenance of equilibrium is the main function of the vestibular apparatus so is vertigo the most important symptom of its involvement. The application of neuro-otology in diagnosing the cause or the location of the cause of vertigo will have a large field of usefulness for there is no branch of medicine to which the symptom of vertigo is foreign. It comes to the ophthalmologist, the syphilologist, the internist, the surgeon and the neurologist.

The ophthalmologist who appreciates the close relationship which exists between the labyrinth and the eye muscles and who appreciates the fact that by these tests any type or direction of nystagmus can be produced will realize the value of these tests in the diagnosis of whether or not a certain eye muscle palsy is a supranuclear lesion. If for example a patient is unable to look to the left and by rotation we can produce a vestibular pull of the eyes in this direction we realize that the pathway between the labyrinth and the eye muscle nuclei is functionating and the lesion must be supranuclear.

We know that the 8th nerve is the most vulnerable to syphilis of all the cranial nerves. Our diagnosis of involvement of this nerve has been made on the subjective examination of the cochlea portion of the nerve and the value of these functional examinations depended largely upon the intelligence and co-operation of the patient. In the great majority of cases the vestibular as well as the cochlea branch is involved and by these tests our findings are gained without the co-operation of the patient. The information is objective and definite and thus accurate. If we can give information of a beginning cerebro-spinal lues, possibly long before other symptoms develop, it may be that we have a method to aid in the prevention of the later manifestations of syphilis of the central nervous system.

To the internist, vertigo has been shrouded in mystery. It has been met with so frequently and in such a variety of conditions and our knowledge of its cause has been so vague that he has been satisfied to speak of it as gastric, hepatic, kidney or cardiac vertigo and if none of these indefinite terms satisfied the conditions it becomes idiopathic vertigo. In the light of our present knowledge of the anatomy and physiology of the vestibular apparatus, we can no longer be satisfied with the use of such general and often meaningless terms in our explanation of the cause of vertigo. Just as a defective vision must be due to some lesion of the ocular tract between the cornea and the occipital lobes, so vertigo must be due to some lesion of the vestibular tract between the semi-circular canals and the temporal lobe. Just as the primary cause of this defective vision may be due to conditions in some other organ so may vertigo be caused primarily by pathological conditions in other parts of the body having a stimulating, impairing or destroying effect upon the vestibular tract. Just as the eyes are examined in certain diseases because of the appearance of a defective vision, so the vestibular tract should be examined when vertigo appears.

To the surgeon these tests will be an aid in that most difficult part of intracranial surgery—exact intracranial localization. Previously in many cases we have had to be satisfied with a palliative decompression. It is possible that by these tests certain lesions will be so localized that something more than a palliative decompression may be done.

On the other hand we know that lesions located in the medulla, pons or cerebellar peduncle, which are, on account of their location, inoperable, often give rise to symptoms suggesting an operable cerebellar lesion. If we can by these tests, so locate a lesion as to be able to state that it is operable or inoperable, we have given to the surgeon a method of examination which will be of great value to him.

Probably to the neurologist more than to any other, will these tests be of the greatest aid. Due to the close relationship between the inner ear and the central nervous system, he is, in his work, often called upon to differentiate between labyrinthine and intracranial lesions. These tests will give definite and absolute information as to whether a lesion is labyrinthine or not. But the value of these tests is not only limited to the differential diagnosis between labyrinthine and intracranial lesions, for often we are able to aid in or to corroborate a location previously made by the neurologist especially if the lesion is located in the cerebello-pontine angle, the cerebellar peduncles, certain parts of the pons or cerebellum.

In regard to the progress of a lesion involving these pathways the information gained by repeated examination is so definite that its value can not be over estimated and these tests are bound to take their place among the routine methods used by the neurologist.

Thus we see that in this examination of the vestibular apparatus our findings are of value in all realms of medicine. But let me impress upon you the fact that these tests are not to take the place of all other methods of diagnosis nor are all pathological conditions to be diagnosed by these tests. The tests are not intended to diagnose the primary cause of vertigo but only the location in the vestibular apparatus of the lesion causing the vertigo. These tests alone will not tell us whether the vertigo is due to a tumor, gumma or abscess of the cerebellum or whether the vertigo is due to a focal infection from the tonsils or teeth or a toxemia from the intestines or kidneys. They will tell us however whether it is a destruction, impairment, or stimulation of the semi-circular canals, or a lesion of the cerebellar peduncle, or the pons, or some other portion of the tract.

If this work is to be of value in diagnosis it must be done by those competent to do it and the methods of examination must be standardized. If one examiner rotates a patient ten times in twenty seconds and with the head bent forward 30 degrees or douches the ear with water 68 degrees, and another rotates the patient ten times in fifteen seconds or uses water at 66 or 70

degrees their results are not going to agree. Those men who have developed this study have worked out on a large number of cases a certain technic which they have learned from experience to be the best. If in our work we find that our results do not agree with them let us, before we decide that these men are wrong and their work valueless, check up carefully our own knowledge and technic.

In conclusion I wish to report a few of the typical cases which have been examined at the University of California Clinic. The histories are brief, only those points of neuro-otological interest are enumerated.

Case No. 1. A. L. K. Private Patient. First seen in April, 1919. Complains of frequent attacks of dizziness since 1913. Attacks begin gradually and not always associated with change of position of head, although he has noticed that during attacks, dizziness is worse on movements of head, attacks of nausea and external objects appear to move back and forth on horizontal line. Staggering with attacks but to no particular side. Noticed a change in hearing about 1916, more marked in left ear. This is increasing. Tinnitus in both ears since 1913, more in left ear. At first intermittent, but last six months more or less continuous. This patient was a rather close observer, and while he gave me a very full and complete history of his condition, I have only mentioned the more salient points. He has noticed that during his vacations, which he spends out of doors tramping and roughing it, he is free of attacks and usually remains so for a couple of months after his return.

Neuro-otological examination—Hearing: diminished in both, much more in right and with 8th nerve involvement. Rotation to right, horizontal nystagmus to left 16 seconds duration. Past pointing: correct direction but shortened. Falling poor. To left: nystagmus to right 15 seconds duration. Past pointing: correct direction but shortened. Falling poor. Caloric: water 68 degrees. Right ear: Rotary nystagmus to left after 110 seconds. Past pointing: correct but shortened. Left ear: Rotary nystagmus to right after 130 seconds. Past pointing: correct but shortened.

Summary—1. Involvement of cochlea portion on both sides.
2. Impairment of vestibular apparatus, both sides.

Diagnosis—Toxic impairment vestibular apparatus, both sides.
3. X-Ray of teeth showed some 5 or 6 root abscesses.

Case No. 2. Robert M. O. P. D. No. 48248. Referred from nerve clinic. Complaint: Dizziness and pain over right parietal region. On February 10, 1918, operated upon in Omaha for acute mastoiditis. Pain in head still continued. February 25, 1918, pus reappeared in ear and mastoid wound. March 10th attack of dizziness and a great difficulty in walking. Mastoid region curetted. On account of continued dizziness, pain and discharge, wound again curetted on April 22, 1918. At present pain on top of head extending to forehead. Very nervous. Dizziness especially at night or in turning around suddenly.

Neuro-otological examination—Right ear at present dry. Hearing: right ear, complete deafness. Left ear: normal. Rotation to right horizontal nystagmus to left 14 seconds duration. Past pointing: correct direction but shortened. Falling poor. To left: horizontal nystagmus to right, 7 seconds duration. Past pointing: none for right arm (repeated examinations), correct but shortened for left arm. Falling fair. Caloric: water 68 degrees. Right ear: no reaction after 4 minutes (repeated examination). Head back: no reaction. Left ear: Rotary nystagmus to right, after 90 seconds. Past pointing: none for right arm; left arm, correct but shortened. Head back: horizontal nystagmus to right good amplitude. Past pointing: none for right arm.

Summary—1. Destruction right labyrinth.
2. At no time was there any past pointing of right arm to left suggesting lesion of inward pointing center of right arm.

Case No. 3. Robert K. Hospital No. 22717. Seen March 21, 1919. Complains of frequent attacks of dizziness beginning in March, 1918. Attacks becoming more frequent and especially noticeable with a change of position of the body. Staggers and falls to left. No trouble with hearing.

Neuro-otological examination—Hearing: normal, both ears. Spontaneous Phenomena: nystagmus, horizontal to right when looking to right. Marked rotary to left when looking to left. Vertical when looking up or down. Past pointing to left with both arms, more marked with left arm. Falling to left: no change with change of position of head. Rotation: to right horizontal nystagmus to left, 42 seconds duration. Past pointing: correct but shortened for right arm, preverted (to left) for left arm. Falling good. To left marked horizontal nystagmus to right, 35 seconds duration. Past pointing: correct direction but exaggerated for both arms. Falling good. Caloric: water 68 degrees. Right ear marked rotary nystagmus to left in 30 seconds. Past pointing: correct for right arm, none for left arm. Head back: horizontal nystagmus to left. Past pointing: correct for right arm, preverted (to left) for left arm. Left ear: marked horizontal (preverted nystagmus to right) in 35 seconds. Past pointing: correct but exaggerated for both arms. Head back: horizontal nystagmus to right. Past pointing correct but exaggerated for both arms.

Summary—1. Normal hearing.
2. Spontaneous nystagmus shows an irritative or active lesion more marked on left side. Vertical nystagmus pathognomonic of brain stem lesion.
3. Spontaneous past pointing of both arms to the left would suggest an irritative lesion in region of inward pointing center of right arm (it is not a destruction of the outward pointing center of right arm, as patient does past point to right with right arm on douching right ear) and a destruction of the inward pointing center for the left arm. This is further corroborated by the fact that the left arm never past points to the right.
4. Prolonged nystagmus on rotation suggests an irritative condition of the fiber of the horizontal canals more marked on left side.

Diagnosis—Multiple lesions of the cerebellum more marked on left side. Lesions are both destructive and irritative.

Comment—Neurological Diagnosis—Multiple Sclerosis.

The following cases No. 4 and No. 5 are particularly interesting as they give an idea of the possible value of these tests. Both cases were referred from the nerve clinic which thus far has been unable to make a diagnosis as there are at present no neurological localizing symptoms. Neuro-otological examination gave a distinct picture of an intracranial lesion which, by these tests is definitely located. If later our diagnosis is confirmed by the neurologist, then the value of these tests as an aid to the neurologist in the location of certain lesions is beyond question.

Case No. 4. Harry S. O. P. D. No. 51616. Age 49. Referred from nerve clinic, April 2, 1919. Complains of dizziness since October, 1918, following attack of Influenza. Attacks becoming more frequent and severe and come on with change of position of head. Staggers at times forward and to the right. Has never fallen. No deafness. Tinnitus intermittent and more in right ear.

Neuro-otological examination—Hearing: good in both ears. Rotation to right: horizontal nystagmus to left, 25 seconds duration. Past pointing: normal. Falling: normal. To left: horizontal nystagmus to right, 30 seconds duration. Past pointing: normal. Falling: normal. Caloric: water 68 degrees. Right ear: faint rotary nystagmus to the left after 80 seconds. Past pointing: correct direction but shortened for both arms. Falling: normal. Head back: 60 degrees horizontal nystagmus to left and past pointing correct for both arms. Left ear: no nystagmus after 4 or 5 minutes douching (2 examinations). Past pointing: correct direction for both arms. Vertigo: normal. Falling: normal. Head back: 60 degrees marked horizontal nystagmus to right, past pointing correct for both arms. Head up and nystagmus disappears.

Summary—1. All reactions go through except nystagmus reaction from left vertical.
2. Right vertical canals pathway slightly impaired shown by prolonged time to produce reaction and shortened past pointing.

Conclusion—Lesion in upper half of pons on left side between the division of the fibers of the left vertical canals and the posterior longitudinal bundle. The slight impairment of the right verticals may be explained by pressure of the lesion on the right pons.

Comment—No diagnosis has yet been made by the neurologist due to the absence of definite localizing neurological symptoms.

Case No. 5. Bosilios S. O. P. D. Referred from nerve clinic. Complains of dizziness since March, 1919. Comes on suddenly and is increasing in severity. Attacks come with change of position of head. Staggers to right. Has fallen to right. Says he has no trouble with hearing and no tinnitus.

Neuro-otological examination—Hearing: complete deafness in right ear (duration unknown). Left ear: normal. Romberg to right, which does not change with position of head. Rotation to right: horizontal nystagmus to left, 15 seconds duration. Past pointing: both arms correct direction. Falling: good. To left: horizontal nystagmus to right, 7 seconds duration. Past pointing: both arms correct. Falling: good. Caloric: water 68 degrees. Right ear: no nystagmus after 4 minutes (two examinations). No past pointing. Head back: no nystagmus or past pointing. Left ear: rotary nystagmus to right, poor amplitude so that difficult to note, after 110 seconds. Past pointing: none for either arm. Head back: marked horizontal nystagmus to right. Past pointing: correct, both arms. Douching left ear with head back 60 degrees and water 68 degrees: marked horizontal nystagmus to right in 27 seconds. Past pointing: correct, both arms. Head up and no nystagmus or past pointing.

Summary—1. Complete destruction of vestibular and cochlea portion of right side.
2. Marked impairment (almost destruction) of fibers from left verticals.
3. Pictures of right cerebello pontine angle lesion of recent origin (not large enough to completely destroy fibers of right verticals as yet).

Comment—No diagnosis as yet by the nerve clinic due to lack of definite localizing symptoms.

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COMPARISON OF THE ACTION OF ROENTGEN RAYS AND RADIUM.*

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In a former paper, the writer described the source and distribution of both radium and X-Rays,[1] and at this time would like to call attention to a comparison of the actinicity of radium and Roentgen rays.

In going over the literature devoted to the physics and therapeutics of radium, one is struck by a confusion of statements as to the penetration and distance traversed by the various radium rays. Also the terms, alpha, beta, and gamma rays, emanation, particles, and waves which are so frequently interchanged that it is difficult even for one familiar with radio physics to intelligently follow the discourse. These terms are often used indiscriminately to designate each and all of the different forms of energy derived from radium.

For the purpose of elucidating the following photographic experiments with radium and Roentgen rays, a few fundamental facts will be reiterated. Radium element in process of decay gives off emanation, an inert gas called niton, and alpha particles, the latter being atoms of positive polarity. The emanation is unstable and loses more alpha particles, after which it is known as radium A. Radium A gives off alpha particles, then resolves itself into radium B, changing in turn to radium C, D, E, and F. The radio active period with which we have mostly to deal is the transition of radium C, for this compound gives off in quantity, alpha, and also the beta and gamma rays, with which we are concerned therapeutically. The alpha rays or particles are not considered seriously in therapeutics. They are of exceedingly limited range of activity and are easily arrested by any filter. If allowed to strike the unprotected skin in quantity, they give rise to a very disagreeable but quite superficial dermatitis. The beta particles are of negative polarity, similar to the Roentgen cathode stream, and in their transmutation give rise to true beta ethereal waves, which the writer believes to be the real therapeutic factors in radium. The gamma radiation is a purely ethereal one of higher penetration and exceedingly short wave length, closely related to the Roentgen ray. The primary beta rays or negative electrons from radium are corpuscular streams, which upon meeting an obstruction are capable of transforming their energy into secondary beta rays, and it is these waves or disturbances that produce the well-known metabolic changes in living protoplasm. The gamma rays are not corpuscular, but at their point of arrest give off secondary rays similar to the beta, and as already stated are of shorter wave length, of relatively higher frequency, and greater penetration. The beta corpuscular stream or primary beta rays

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